**R. James Cook**
Agricultural Research, Science and Education Administration, U.S. Department of Agriculture, Pullman, WA

# Fusarium Foot Rot of Wheat and Its Control in the Pacific Northwest

Control of soilborne pathogens is one of the greatest challenges facing agriculture. Soil fumigants can be effective but the cost is prohibitive except with high-value crops. Breeding for host plant resistance has been ineffective for most soilborne pathogens, in part because of unreliable methods to test for resistance and in part because soilborne plant pathogens are commonly unspecialized in their host range so that effective varietal resistance is not available.

Traditionally, soilborne pathogens have been held in check in agricultural soils by cultural practices, including long rotations, clean tillage (even burning of infested residue in some cases), and use of organic fertilizers. In the United States and many other countries, these practices are being replaced with short-term rotation or no rotation (monoculture), less or no tillage, and inorganic fertilizers. In addition, the trend toward more intense crop management to increase productivity may be predisposing some crops to previously unimportant soilborne pathogens.

Soilborne plant pathogens are still controlled in some areas and on some farms by long rotations, clean tillage, and organic amendments; and by managing the crop at a level lower than needed to obtain full yield potential. More and more, however, farms must be specialized and management intensified as a matter of economic survival. The control of Fusarium foot rot of wheat illustrates how knowledge of the ecology of a pathogen, together with an understanding of a stress factor in the crop, can open the way to controlling a soilborne, stress-related pathogen without lowering the yield potential of the crop or significantly changing the method of farming.

**Fig. 1. Wheat tillers with foot rot caused by *Fusarium roseum* 'Culmorum.' Leaf sheaths have been stripped away to reveal diseased culms.**

**Fig. 2. Wheat plant of the cultivar Nugaines with premature blight caused by *Fusarium roseum* 'Culmorum.'**

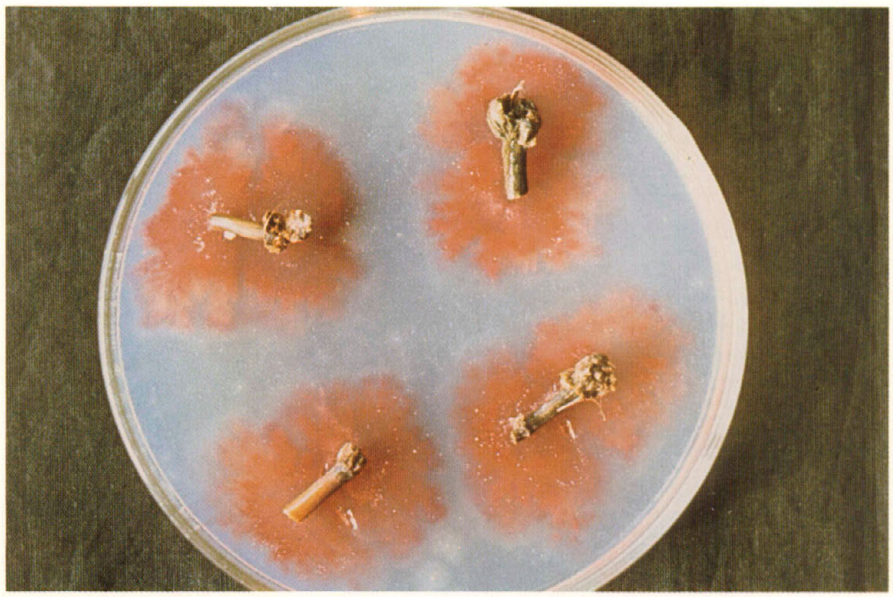

**Fig. 3. Typical colonies of *Fusarium roseum* 'Culmorum' around infected nodal segments of wheat culms with foot rot and cultured on acidified corn meal agar.**

## The Disease in the Pacific Northwest

The Fusarium foot rot considered in this article occurs mainly on wheat but also on barley and oats in the Pacific Northwest (3). The disease is caused by either *Fusarium roseum* Link emend. Snyd. & Hans. 'Culmorum' (= *F. culmorum* W. G. Smith) or *F. roseum* 'Graminearum' (= *F. graminearum* Schwabe). The symptoms caused by these two closely related fungi are nearly identical (3), but since Culmorum is the main cause of Fusarium foot rot in the northwestern United States, most of the information in this article is based on studies of Culmorum. Graminearum is limited to the warmest portions of the region (eg, south central Washington),

whereas Culmorum is widely distributed in central and eastern Washington, north central Oregon, and northern Idaho.

Fusarium foot rot is characterized by a decay of the crown and basal stem tissues (Fig. 1), thus the name. Infected plants rarely show outward symptoms until after heading, by which time the foot rot is sufficiently advanced to prevent water transport. Entire plants die prematurely (premature blight), resulting in "white heads" (Fig. 2).

The primary inoculum is soilborne chlamydospores and infested plant debris contained mainly in the surface 10 cm of soil (mulched layer). Culmorum and Graminearum both form thick-walled chlamydospores that are morphologically indistinguishable (12). Chlamydospores of Graminearum are more likely to die under conditions of high soil temperature and rapid desiccation than are those of Culmorum. This is anomalous, since the optimal temperature for mycelial growth is higher and the optimal water potential is as low as or lower (drier) for Graminearum than for Culmorum (7). Moreover, the average air temperature in July is 3–4 C higher in areas where Graminearum is important than where Culmorum is important (12). The occurrence of foot rot caused by a particular *Fusarium* is thus correlated with the temperature and water potential most appropriate for mycelial growth of the fungus and not with conditions best suited for survival of chlamydospores. The net result is that chlamydospores of Graminearum are rare or nonexistent (do not persist) in the traditional Graminearum areas of the Pacific Northwest. Mycelium in crop residue is the main form of inoculum of this pathogen (12,14).

Entry into the crown is gained approximately 2–3 cm below the soil surface either through openings around emerging crown (secondary) roots or by infection of the newly emerging crown roots. The dry soil conditions occurring in the top 10 cm of soil in the fall in the Northwest are ideal for infection. From the infected crown, and depending on the degree of host stress, the pathogen may progress up the culm one to three internodes during or shortly after heading. Outer leaf sheaths enclosing the infected internodes remain symptomless and apparently even pathogen-free, at least in the Pacific Northwest, where relative humidity is low within the plant canopy during the period of greatest foot rot development.

Diseased internodes become chocolate brown in color (Fig. 1), which can be observed by stripping away the usually bright, healthy-appearing sheaths wrapped around the internodes. Abundant mycelium, often pink or burgundy colored, can be observed within the hollow stem by splitting a diseased internode longitudinally. The spongy nature and brown color of the diseased crown can be observed by cutting the crown longitudinally.

## Isolating the Pathogen from Tissue and Soil

To isolate and identify these pathogens from diseased tissue, I use acidified Difco corn meal agar (2 ml of 25% lactic acid per liter of molten medium added after autoclaving). Crown tissue or internodal segments with sheath removed are washed about 1 hour under running tap water, surface-sterilized for 10–15 seconds in 0.5% sodium hypochlorite, and then plated four pieces per 9-cm-diameter petri dish. The dishes are incubated in the dark for 7–10 days at about 10 C, then moved to a table in a room with natural daylight and a temperature of 20–25 C to promote sporulation. *Fusarium* develops diagnostic colonies around each piece (Fig. 3), and the taxon can be identified according to morphology of the macroconidia (5).

Dilution plates of soil suspensions can be used to estimate populations of Culmorum and confirm a diagnosis. The *Fusarium*-selective medium of Nash and Snyder (9) is ideal for this. The medium contains 1.5% Difco peptone, 2% agar, 0.1% $KH_2PO_4$, 0.05% $MgSO_4 \cdot 7H_2O$, 300 ppm streptomycin as streptomycin sulfate, and 0.1% pentachloronitrobenzene (PCNB) as a 75% wettable powder. Soil from a field with a high incidence of suspected foot rot caused by Culmorum should have several hundred or even 1,000–10,000 Culmorum propagules per gram. If only a trace population of Culmorum is detectable by dilution plating of soil (eg, 10–50 propagules per gram), the visual diagnosis should be reevaluated. Conditions sometimes mistaken for Fusarium foot rot include frost damage of the lower internodes, severe Cephalosporium stripe, and take-all before the typical blackening develops.

## Other Wheat Diseases Caused by *Fusarium roseum*

Head blight, scab, seedling blight, and root rot are other manifestations of an attack on wheat by *Fusarium* and should not be confused with foot rot. Head blight and scab occur in humid climates where the primary inoculum comes from either airborne ascospores or water-splashed conidia deposited directly in or among the spikelets of heads, usually during flowering. Seedling blight is a problem mainly in areas with scab, ie, where the *Fusarium* is seedborne. Root rot caused by *Fusarium* occurs on wheat as brown or reddish-brown lesions extending a few millimeters to 1 cm or more along the root axis. The disease, referred to as common root rot and prevalent in the Great Plains of the United States and the prairie provinces of Canada, is caused by *Fusarium* spp. together with *Helminthosporium sativum* (*Bipolaris sorokiniana*). A root rot caused by *Fusarium* can lead to foot rot in wheat, but the succession is not automatic and the two disease phases should be recognized as distinct.

At least five closely related but distinct taxons within *F. roseum* can cause one or more of the several manifestations of a *Fusarium* attack on wheat (5), depending on conditions. *F. roseum* 'Graminearum' contains two groups (I and II) based on differences in life cycles and ecological requirements (8). The Graminearum population responsible for foot rot in the Pacific Northwest and in Queensland, Australia, (2) belongs in Group I. The other three taxons include *F. roseum* 'Crookwell' (1), *F. roseum* 'Avenaceum,' and *F. roseum* 'Culmorum.' Of these five taxons (which many *Fusarium* specialists prefer to recognize as three or more species rather than as all members of *F. roseum*), only Graminearum Group I, Culmorum, and Avenaceum have been found in the Pacific Northwest. Avenaceum may cause scab on wheat in high rainfall areas west of the Cascade Mountains and it occurs in dryland areas east of the Cascades, but I have not found this fungus associated with foot rot of wheat in the Pacific Northwest.

## Onset of Severe Foot Rot in the Pacific Northwest

Fusarium foot rot under Pacific Northwest conditions occurs mainly in low to intermediate rainfall (20–40 cm annual precipitation) areas on dryland cereal grains (chiefly wheat) grown on fallow (Fig. 4). With the wheat-fallow management, a field harvested in late July or early August of one year is not planted again until late August or early September of the next year. By this method, one crop is produced on the net accumulation of precipitation for 2 years (11).

Although *Fusarium* has long been recognized to cause root rot of wheat in the Pacific Northwest (13), the first outbreaks of severe foot rot in the region were observed in 1964 by G. W. Bruehl, C. S. Holton, and O. A. Vogel. Two fields in east central Washington were severely damaged in 1964, one near Ritzville and the other near Harrington. Both fields were in an area with average annual precipitation of 25 cm and both were planted to the potentially high-yielding, semidwarf soft white wheat cultivar Gaines. This was the first semidwarf wheat cultivar grown in the Pacific Northwest and had been released only 3 years earlier, in 1961. Culmorum was the pathogen in both cases. Dilution plate counts of soil from these two fields in 1965 revealed about 3,000 propagules of Culmorum per gram of soil in the Ritzville field and 1,500 propagules of Culmorum per gram of soil in the Harrington field. In general, 100

propagules of Culmorum per gram of soil is sufficient to cause damage if conditions for disease are favorable (3).

A field of Gaines wheat near Pullman, Washington, on Palouse silt loam in the higher rainfall area (50 cm annual precipitation) was severely damaged by Fusarium foot rot in 1966. The Culmorum population was nearly 2,000 propagules per gram, based on dilution plate counts of soil removed in July, just before harvest of the affected crop. Unexpectedly, the Culmorum population disappeared from the Pullman field within 12 months (by late 1967), in contrast to the two fields in the drier area with Ritzville silt loam, where the propagule count fluctuated above 1,000 per gram for season after season and today is still several hundred per gram, even though Fusarium foot rot has not been evident in these fields for the past four or five crops. Subsequent observations have suggested further that Culmorum is short-lived in the Palouse silt loam of the Pullman area and perhaps in southeastern Washington generally. The reason for the decline of the Culmorum population in the Palouse field but not in the fields farther west remains a mystery.

A population of Culmorum may increase in at least two ways: 1) chlamydospores formed in mycelium in or near host debris are released into the soil as the debris decomposes and 2) chlamydospores are formed within macroconidia when the latter, from sporodochia on diseased culms or colonized debris, become mixed into the soil. Mycelium in host debris provides a mechanism for steady maintenance of or gradual increase in the population, whereas macroconidia can result in population increases of severalfold in a single season.

Asexual sporulation of Culmorum is especially profuse on parasitized stems of oats (4). The Ritzville field, with 3,000 propagules of Culmorum per gram of soil in 1965, had been planted to spring oats in 1962 (with fallow in 1963). An adjacent field planted to spring wheat in 1962 had only 300 propagules of Culmorum per gram of soil in 1965. Similarly, with the Pullman field the area with the high Culmorum population in 1966 corresponded exactly to the area planted to oats in 1966 (Fig. 5). Further studies confirmed that parasitized oat culms support more rapid asexual sporulation than do parasitized wheat culms (4).

Host tissues are colonized by mycelium of Culmorum mainly through parasitic activity of the fungus (6). Culmorum is a highly competitive saprophyte but is preempted as a saprophytic colonist of wheat straw in soil by common, airborne saprophytic fungi that become established in the straw after crop maturity but before tillage. Thus, Culmorum is limited in occupancy of straw almost exclusively to that colonized through parasitism.

## Role of Plant Water Stress

The connection between plant water stress and development of *Fusarium* foot rot of wheat was first indicated by the following observations:

- The Pullman field with severe Fusarium foot rot in 1966 consisted mainly of a south-facing, 30° slope with a low-lying flat on one side (Fig. 5). Fusarium foot rot was severe (most plants died prematurely) on the slope but was mild or nonexistent (nearly all plants were a healthy green) in the flat. The transition zone between diseased and healthy plants was very sharp (Fig. 5). Dilution plate counts of the Culmorum population in soil samples taken on parallel lines 1–2 m below and 1–2 m above the transition zone revealed nearly identical populations: 1,900–2,000 propagules per gram. The only apparent difference was the plant stress on the slope but not in the flat. This observation revealed that a high population of the pathogen does not insure a high incidence of disease.
- Wheat planted in early September 1967 in the Ritzville field was nearly 100% infected (the pathogen could be recovered from surface-sterilized pieces of crown tissues representing 128/130 random plants) 12 weeks later, yet Fusarium foot rot with premature blight did not develop as the plants matured the following spring and summer. Rainfall in June (immediately after the crop had headed) greatly reduced the potential for plant water stress. This observation revealed that crown infection with *Fusarium* does not insure a high incidence of foot rot.
- In the Harrington field, nitrogen is applied as anhydrous ammonia injected at the time of planting; the seed is planted in rows 40 cm apart and a shank is positioned to inject anhydrous ammonia between every two rows (80 cm apart). If for any reason a shank fails to conduct ammonia, two rows are left to grow with only residual nitrogen in the soil. When this happened in 1970, the rows starved of nitrogen showed no evidence of Fusarium foot rot in spite of the high incidence of

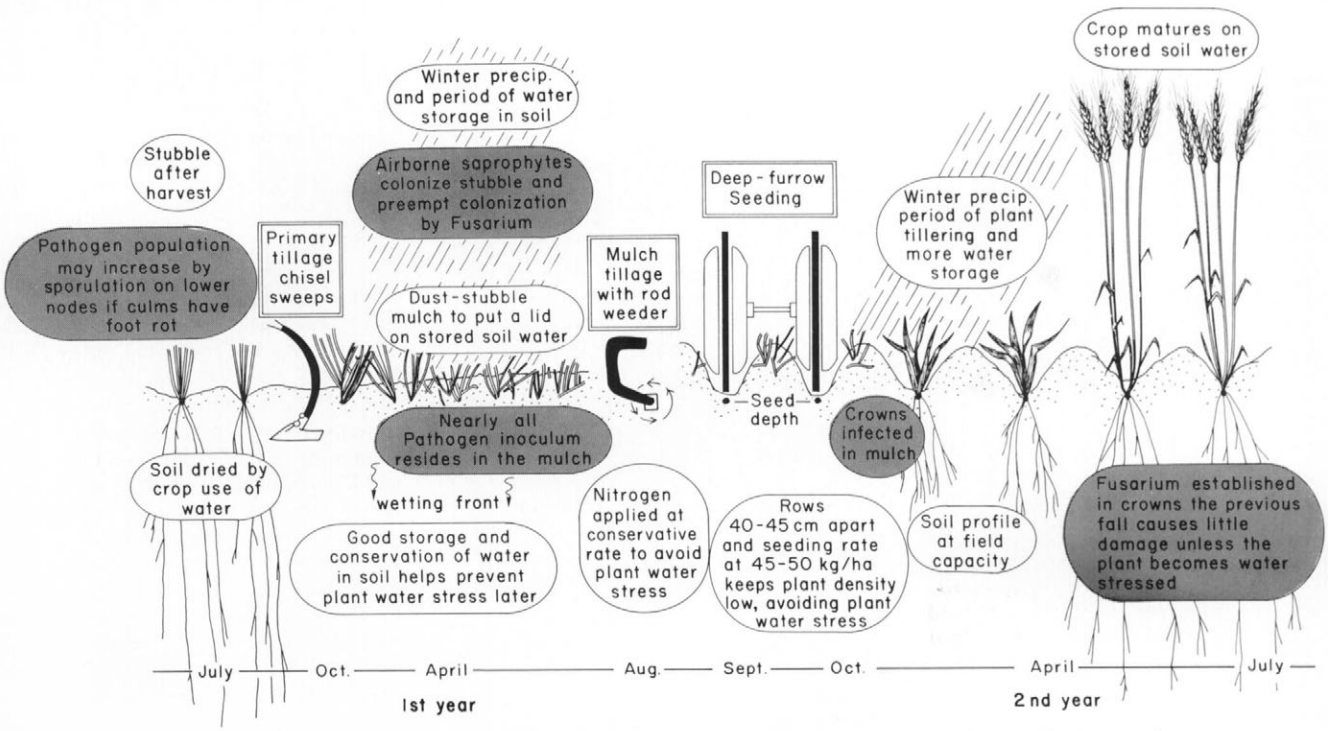


**Fig. 4. How the wheat-fallow cycle in the Pacific Northwest controls foot rot caused by *Fusarium roseum* 'Culmorum.'**

the disease in adjacent rows (Fig. 6). This observation suggested that severity of Fusarium foot rot could be reduced by carefully managing the rate of nitrogen application.

R. I. Papendick and I (10) demonstrated that the higher the rate of nitrogen fertilizer applied, the lower the midday leaf water potential beginning in late May or early June (plants in the late boot stage or headed). Water potential is an expression of the energy status of the water; the lower the plant water potential, the greater the stress. In our studies, plants in plots with rows 30 cm apart and receiving high rates of nitrogen (100–200 kg/ha) developed midday leaf water potentials approaching −40 bars. In contrast, plants in plots with the same row spacing but receiving low rates of nitrogen (none or 50 kg/ha) developed midday leaf water potentials no lower than −30 to −35 bars and commonly were above −30 bars until normal maturity. The low water potentials with high nitrogen resulted from the greater leaf area associated with the higher rates of nitrogen and consequently the greater loss of soil water per unit area of field because of transpiration. When the distance between rows was increased to 60 and then to 90 cm, midday leaf water potentials were lowered accordingly and Fusarium foot rot was reduced, even with high rates of nitrogen. The wide-row spacing resulted in fewer plants and hence a slower rate of soil water use per unit area of field.

**Fig. 5. Field of the winter wheat cultivar Gaines near Pullman, Washington, in 1966 with damage from foot rot caused by *Fusarium roseum* 'Culmorum.' The pathogen population was about 2,000 propagules per gram of soil slightly more than half way up the slope but was essentially undetectable on the hilltop (note demarcation line). The high infestation of *Fusarium* resulted from oats grown the previous year on the lower portion of the field. The pathogen population was about 2,000 propagules per gram of soil where the wheat was green at the lower edge of the field (just beyond the road), but Fusarium foot rot did not develop, apparently because the plants did not develop water stress.**

**Fig. 6. Nugaines wheat seeded by deep-furrow drill in rows 40 cm apart; nitrogen was applied as anhydrous ammonia through a shank between every other pair of rows. The middle pair of rows received no nitrogen and consequently shows no "white heads" from foot rot caused by *Fusarium roseum* 'Culmorum.'**

To obtain further evidence that water stress and not nitrogen per se was the predisposing factor to Fusarium foot rot, we established replicated plots on the Washington State University Experiment Station at Lind, where an irrigation treatment could be applied. The plot site was infested with approximately 3,000 Culmorum propagules per gram of soil to a depth of 10 cm. The treatments included high and low nitrogen with and without irrigation; irrigation was applied once in May and again in June. Direct isolation from crowns of random plants removed in April revealed more than 50% plant infection before the irrigation treatment was imposed. Midday leaf water potentials were low (plants stressed) only in the plots with high nitrogen and no irrigation. This treatment also resulted in Fusarium foot rot (Table 1). Where water was applied, leaf water potentials remained high (plants not stressed) and *Fusarium*, already in the crowns of plants by April, did not advance to produce severe foot rot.

Fusarium foot rot does not develop readily on wheat in greenhouse pots, a situation that has restricted our research entirely to the field. Because plants in pots must be watered frequently, low plant water potentials are difficult or impossible to maintain. R. I. Papendick and I (*unpublished*) grew wheat in 40-L galvanized cans 35 cm in diameter and 40 cm deep, with 10 plants per can. Water was supplied sparingly and only to soil at the bottom of the can, and the top 20–25 cm of soil was nearly air dry during plant heading and maturity. Even with this treatment, headed plants never developed midday leaf water potentials below −24 bars. In the field, we would not expect Fusarium foot rot to develop in plants

with water potentials no lower than this. Little wonder we cannot produce Fusarium foot rot in the greenhouse.

Low plant water potentials sustained during the later stages of plant development are clearly a key to the development of Fusarium foot rot. Without the predisposing plant water stress, plants may have root rot and even mild infections of the crown but are unlikely to develop severe foot rot. The stage of plant growth at the time of stress may be equally important, but we have no information on this factor.

Wheat cultivars differ in depth of rooting, leaf area, number of stomata per leaf, and other factors important to rate of transpiration and rate of soil water use. Cultivars also vary in susceptibility to Fusarium foot rot, with disease reaction depending at least partially on the stress pattern for that particular genotype. Without exception, wheat cultivars able to maintain high leaf water potentials after heading (ie, least likely to stress) show the least Fusarium foot rot. Several club wheat cultivars (eg, Elgin, Omar, Paha, and Moro) are less likely to stress than several common semidwarf white wheats, including Gaines, Nugaines, Hyslop, and McDermid. The common semidwarf white cultivar Sprague also has less tendency to stress—and develops less Fusarium foot rot. An exception is Luke, a common semidwarf white wheat that develops water potentials as low as or lower than those of Gaines or Nugaines yet is less prone to develop severe Fusarium foot rot than are Gaines and Nugaines. More work is needed to sort out the many possible factors in water stress, in resistance to Fusarium foot rot, and in combinations of these. Meanwhile, control of Fusarium foot rot is now possible by selecting the proper cultivar and using management practices that delay plant water stress.

**R. James Cook**

Dr. Cook is a research plant pathologist in the Regional Cereal Disease Research Laboratory, U.S. Department of Agriculture, Science and Education Administration, Agricultural Research, Pullman, WA. He obtained B.S. and M.S. degrees from North Dakota State University and a Ph.D. from the University of California, Berkeley, in 1964. He was a NATO Post-doctoral Fellow to the Waite Agricultural Research Institute, Adelaide, Australia, during 1964–1965 and a Guggenheim Fellow to the CSIRO Division of Soils, Adelaide, Australia, during 1973–1974. His research is on the biology and control of soilborne pathogens of Pacific Northwest wheat. Besides his research on soil-water relations in plant disease, he has conducted studies on biological control of soilborne plant pathogens and authored with K. F. Baker the book "Biological Control of Plant Pathogens," published in 1974.

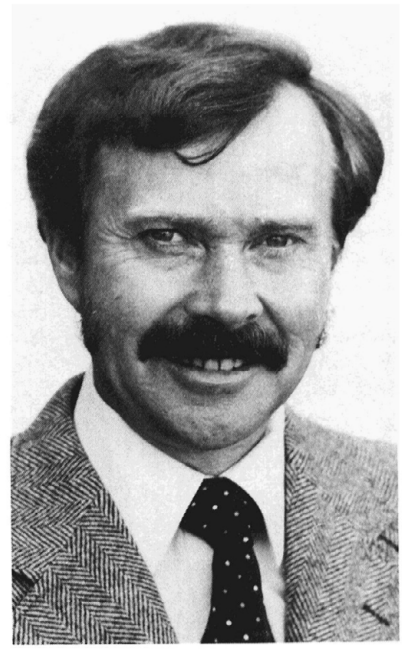

**Table 1.** Influence of irrigation and nitrogen on development of foot rot caused by *Fusarium roseum* 'Culmorum' in wheat plants[a]

| Treatment[b] | Tillers per foot rot category (%)[c] | | |
|---|---|---|---|
| | Healthy | Mild | Severe |
| Irrigated | | | |
| High nitrogen | 89.4 | 10.2 | 0.5 |
| Low nitrogen | 86.3 | 12.9 | 0.9 |
| Not irrigated | | | |
| High nitrogen | 40.7 | 45.8 | 13.4 |
| Low nitrogen | 54.3 | 38.8 | 6.8 |

[a] The experimental site contained about 3,000 propagules of the pathogen per gram of the top 10 cm of soil.
[b] Irrigation treatment was application of water (8–10 cm) in May and again in June; plants were infected before irrigation. High nitrogen = about 150 kg/ha applied at seeding; low nitrogen = none applied, residual only.
[c] Healthy = no foot rot evident; mild = crown decayed, with discoloration only 1–2 cm up the culm; severe = typical symptoms of acute foot rot, with brown discoloration two to three internodes up the culm.

## Specific Management Practices

The practices currently in use or recommended for control of Fusarium foot rot in dryland wheat in the Pacific Northwest are mainly those minimizing population increases of the pathogen and reducing or delaying onset of water stress in the host (Fig. 4). Specifically, the practices are:

1. Since oats support the greatest amount of asexual sporulation and are thus most likely to cause rapid increases in the population of Culmorum, growers avoid this crop in their otherwise wheat-fallow or barley-fallow rotations.

2. After harvest but before the rainy season begins, the field with standing stubble is worked with a chisel plow to improve infiltration and reduce water runoff during precipitation or snow melt. This tillage also helps control weeds that otherwise would remove soil water.

3. In the spring, a dust and stubble mulch is established with tillage using a rod weeder. This mulch is about 10 cm deep and protects against water evaporation from the fallow during the hot summer months (11). In addition, the straw kept on the soil surface as part of the mulch becomes thoroughly colonized by airborne saprophytic fungi and thus is unavailable to Culmorum as a saprophytic colonist in soil.

4. The rate of application of nitrogen as anhydrous ammonia is based on a soil test for residual nitrogen but probably will not exceed 60–75 kg/ha in an area with 25 cm average annual precipitation.

5. The field is planted in early September. Seeding in late August results in large plants and ultimately greater water stress. Later seeding (eg, late September or early October) lowers the yield potential. The early September seeding provides the best compromise between maximum yield potential and minimum water stress potential. Virtually all growers now use a deep-furrow drill to plant seed in moist soil beneath the dust mulch. Rows are 40–45 cm apart and the seeding rate is 45–50 kg/ha. A wider row spacing would be even better but favors weed growth.

When all these practices have been used, control of Fusarium foot rot of dryland wheat on fallow in the Pacific Northwest has been excellent, even in fields with a high infestation of Culmorum. For example, in the Harrington field, Gaines wheat yielded about 2,800, 2,557, and 2,933 kg/ha in 1964, 1966, and 1968, respectively, with nitrogen rates of 106, 52, and 100 kg/ha in those 3 years (Robert Kramer, Harrington, WA, *personal communication*). In 1970, Unitan (spring) barley was

grown in the field, with a nitrogen rate of about 47 kg/ha, and the yield was 2,695 kg/ha. The field was then seeded to Luke wheat in early September 1971, at which time the Culmorum population in 30 random soil samples averaged 4,500 propagules per gram, the highest population ever recorded for this field. Anhydrous ammonia was applied at seeding in the fall of 1971 to give 70 kg of nitrogen per hectare. The crop showed only a trace of foot rot and yielded 3,060 kg/ha. In experimental plots of Nugaines in the same field that same year, about 30% of the tillers had foot rot where nitrogen was applied at 67 and 130 kg/ha. The field was planted to Moro with 60 kg of nitrogen per hectare in 1974, half to Luke and half to Sprague with 70 kg/ha in 1976, entirely to Luke with 70 kg/ha in 1978, and entirely to Daws (common semidwarf soft white) with 70 kg/ha in 1980. The yields for these crops were, respectively, 4,262, 3,084, 3,811, 2,868, and 4,664 kg/ha. Thus, the five crops since 1972 averaged nearly 1,000 kg/ha (14 bu/A) more than the Gaines crops in 1964, 1966, and 1968 while receiving an average of 18 kg less nitrogen per hectare per crop. A significant part of the increase in yield and in nitrogen efficiency is due to Fusarium foot rot control, although other reasons cannot be ruled out. For example, the high yield of Daws in 1980 resulted mainly from 23% above-normal rainfall in June 1980.

The practices I have described to control Fusarium foot rot of dryland wheat in the Northwest should not be expected to work against other kinds of damage by *Fusarium*, such as scab, head blight, seedling blight, or possibly even common root rot. Each problem is unique and requires a different approach.

## Literature Cited


1. BURGESS, L. W., P. E. NELSON, and T. A. TOUSSOUN. 1980. Characteristics of a newly recognized population, *Fusarium roseum* 'Crookwell.' Trans. Br. Mycol. Soc. In press.
2. BURGESS, L. W., A. H. WEARING, and T. A. TOUSSOUN. 1975. Surveys of Fusaria associated with crown rot of wheat in eastern Australia. Aust. J. Agric. Res. 26:791-799.
3. COOK, R. J. 1968. Fusarium root and foot rot of cereals in the Pacific Northwest. Phytopathology 58:127-131.
4. COOK, R. J. 1968. Influence of oats on soil-borne populations of *Fusarium roseum* f. sp. *cerealis* 'Culmorum.' Phytopathology 58:957-960.
5. COOK, R. J. 1981. Fusarium diseases of wheat and other small grains in North America. In: P. E. Nelson, T. A. Toussoun, and R. J. Cook, eds. The Genus, *Fusarium*. Pennsylvania State University Press, University Park. In press.
6. COOK, R. J., and G. W. BRUEHL. 1968. Relative significance of parasitism versus saprophytism in colonization of wheat straw by *Fusarium roseum* 'Culmorum' in the field. Phytopathology 58:306-308.
7. COOK, R. J., and A. A. CHRISTEN. 1976. Growth of cereal root-rot fungi as affected by temperature-water potential interactions. Phytopathology 66:193-197.
8. FRANCIS, R. G., and L. W. BURGESS. 1977. Characteristics of two populations of *Fusarium roseum* 'Graminearum' in eastern Australia. Trans. Br. Mycol. Soc. 68:421-427.
9. NASH, S. N., and W. C. SNYDER. 1962. Quantitative estimations by plate counts of propagules of the bean root rot *Fusarium* in field soils. Phytopathology 52:567-572.
10. PAPENDICK, R. I., and R. J. COOK. 1974. Plant water stress and development of Fusarium foot rot in wheat subjected to different cultural practices. Phytopathology 64:358-363.
11. PAPENDICK, R. I., M. J. LINSTROM, and V. L. COCHRAN. 1973. Soil mulch effects on seedbed temperature and water during fallow in eastern Washington. Proc. Soil Sci. Soc. Am. 37:307-314.
12. SITTON, J. W., and R. J. COOK. 1981. Comparative morphology and survival ability of chlamydospores of *Fusarium roseum* 'Culmorum' and 'Graminearum.' Phytopathology. In press.
13. SPRAGUE, R. 1950. Diseases of Cereals and Grasses in North America. Ronald Press Co., New York. 538 pp.
14. WEARING, A. H., and L. W. BURGESS. 1977. Distribution of *Fusarium roseum* 'Graminearum' Group I and its mode of survival in eastern Australian wheat belt soils. Trans. Br. Mycol. Soc. 69:429-442.